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ESTIMATING THE CONTRIBUTION OF LEAD-BASED PAINT TO SOIL LEAD, DUST LEAD, AND CHILDHOOD BLOOD LEAD

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ABSTRACT: Young children are particularly susceptible to lead carried in fine particles of surface soil (exterior dust) and in household dust. Multi-media environmental and biological samples may allow causal inferences about the relative importance of different sources and pathways in childhood lead exposure, and about the effectiveness of intervention strategies. Methods include: (1) statistical inferences about pathways using structural equation modeling; (2) inferences about pathways using physical tracers of sources; (3) inferences based on mass balance estimates.

Structural equation modeling allows estimation of both direct and indirect exposures to lead-based paint. For example, chips of exterior lead-based paint may be ingested directly, may contribute to surface soil lead which is ingested, and may also be transported into household dust which is ingested. It is often possible to identify soil and dust exposure from elevated lead levels on the child's hands. We use structural equation models in cross-sectional field studies in some Western communities, to demonstrate the role of lead paint as a source of lead exposure. Stronger causal inferences about sources and pathways can be made when there are physical identifiers of the source, such as an unusual ratio of some stable lead isotopes. Another approach is to carry out an intervention, such as removal or encapsulation of the lead-based paint, or removal of the contaminated soil and dust associated with the paint. If lead-based paint is not removed and there is some recontamination of these media over time after the intervention, then we can attribute the exposure to lead paint.

KEYWORDS: lead, soil lead, dust lead, lead-based paint, hand lead, blood lead, environmental pathways, exposure assessment, structural equation model, nonlinear regression, repeated measures analysis, stable lead isotope, lead tracer, dust mass transport, lead abatement.

INTRODUCTION

Any particular source of environmental lead may have several pathways leading to undue exposure for a child or for other targets. Identification and quantification of these pathways may be very helpful in determining the most effective and/or cost-effective interventions or abatements for preventing childhood lead exposure. Young children are especially vulnerable to excessive lead exposure in the home. Because of their size and activity, they are likely to inhale more air and consume more tap water per body weight than are adults. Young children are particularly susceptible to ingesting lead carried in fine particles of surface soil (exterior dust) and in household dust particles. Fine particles adhere to the child's hands and skin and are swallowed during the course of normal childhood hand-to-mouth activity. Charney et al. [1] have shown that effective

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control of household dust was sufficient to keep blood lead levels of heavily lead-burdened children from increasing to pre-treatment levels when they returned to the same residential unit after treatment. Blood lead levels in treated children were still high, possibly from other exposure media inside or outside the home, but also from the large burden of lead stored in the child's skeleton. Rosen et al. [2] have shown that endogenous lead sources in a child's cortical bone are only partially removed by chelation therapy, and decrease very slowly after environmental abatement without chelation. There is thus a built-in limitation in the effectiveness of lead abatement in reducing blood lead levels or body lead burdens of lead-exposed children. It is clearly preferable to prevent exposure by removing the source, or by preventing exposure to media contaminated by that lead source.

Lead-based paint (abbreviated as LBP in this paper) may be available to the child in several media: as chips or flakes of deteriorating LBP, chewed directly from the painted surface; as a contributor to lead contamination of the surface soil near a LBP painted surface; as a contributor to dust in window wells, on window sills, and at entry areas; and as a contributor to household dust, both from exterior LBP to soil to dust and from interior LBP to interior dust. When sufficient data are available to characterize the lead levels in all relevant exposure media, then it may be possible to attribute certain fractions of the total exposure to differences in lead levels in the various media, and thence to a primary source of the exposure such as LBP. Several physical and statistical methods have been used to do this. We will show how these methods may be applied when there is enough information to adequately define the child's total lead exposure.

PHYSICAL IDENTIFICATION OF ENVIRONMENTAL LEAD PATHWAYS

MASS BALANCE ESTIMATES

A few simple calculations will demonstrate that lead-based paint can make significant contributions to household dust and to exterior soil near the house. The amount of lead-based paint on walls can be estimated from the HUD National Hazard Survey [3]. The area covered by LBP depends on the age of the house. The average interior LBP area on houses with LBP is 157 square feet for houses built between 1960 and 1979, 463 square feet for houses built between 1940 and 1959, and 1250 square feet for houses built before 1940. The average exterior LBP area on houses with LBP is 521 square feet for houses built between 1960 and 1979, 851 square feet for houses built between 1940 and 1959, and 1384 square feet for houses built before 1940. (One . $ft^2 = 0.0929\ m^2$.) The average XRF levels also depend on the age of the house, with a geometric mean XRF of 0.59 mg Pb/cm^2 for houses built between 1960 and 1979, 0.70 mg Pb/cm^2 for houses built between 1940 and 1959, and 1.1 mg Pb/cm^2 for houses built before 1940. One may thus estimate the amount of lead on a typical (roughly, median) house built between 1960 and 1979 as 0.1 kg inside and 0.4 kg outside, for houses built between 1940 and 1959 as 0.3 kg inside and 0.6 kg outside, and for houses built before 1940 as 1.3 kg inside and 1.4 kg outside. Thus, for analysis purposes, we may take about 1 kg lead paint each on the interior and the exterior.

Lead-based paint deteriorates slowly over time, by design. We may assume that a typical lead-painted interior surface (about 1 mg Pb/cm^2) will lose all of its paint in, say, 100 years, so that the rate of accumulation of lead from interior LBP in household dust may be about 0.01 kg Pb/year.

The typical amount of household dust in older housing is about 5 kg. For example, in the Baltimore houses selected for the Three-City Study, the mean dust loading is about 49 g/m^2. With a typical floor area of about 1100 ft^2 (100 m^2) the estimated total dust content is about 4900 g = 4.9 kg in these houses.

The contribution of interior LBP to this dust load depends on the rate of removal of leaded dust (presumably the same as the rate of removal of total dust) from the house. Farfel et al. [4,5] found that there was significant dust recontamination of Baltimore houses after LBP abatement by traditional or modified abatement methods, with a first-order time constant that we have estimated as about 2 to 3 months. Allott et al. [6] showed that the rate of removal of the radioactive isotope ^{137}Cs in dust from British homes following the Chernobyl explosion was closely exponential with a time constant of 9 to 12 months. We assume a rapid rate of dust removal, at a rate of 4 times per year (time constant 0.25 years). The steady-state level of lead in floor dust from LBP is therefore equal to (0.01 kg Pb/year) * 0.25 years = 0.0025 kg. The steady-state contribution of interior LBP to dust lead concentration is 0.0025 kg Pb / 5 kg dust = 500 μg Pb /g dust. Roughly similar levels may be expected to characterize LBP contributions to lead in surface soil near the house perimeter or dripline. These levels are very similar to those we have observed in a number of studies.

USE OF STABLE LEAD ISOTOPES TO IDENTIFY PATHWAYS

Several studies have use the isotopic composition of lead to identify source contributions. While ^{208}Pb is the most common isotope, smaller quantities of ^{207}Pb, ^{206}Pb, and ^{204}Pb characterize lead with different radiogenic or cosmogenic origin. Lead added to gasoline or paint during manufacture will usually come from a single location, such as Broken Hill in Australia or from the Missouri Lead Belt, will have different ratios of these isotopes. Yaffee et al. [7] studied 12 children in Oakland, CA in 1978. The children lived in 2 dwelling units. The authors conclude that "the isotopic ratios of lead in the blood of these children were close to the average lead ratios of paints from exterior walls and to the lead ratios of surface soils in adjacent areas where the children played. In both case studies, the data suggest that the lead in soil was derived mainly from the weathering of lead-based exterior paints and that the lead-contaminated soil was a proximate source of lead in the blood of the children." The children were not pica-prone and probably did not ingest large paint chips directly, since the blood lead levels were not excessively elevated (maximum 43 μg/dl). The children spent much time playing in the lead-contaminated soil (1050 to 1370 μg Pb/g soil). Indoor dust samples were collected and had isotopic compositions similar to soil lead and to paint, but higher lead concentrations (1200 to 3300 μg Pb/g dust). The total exposure to lead derived from LBP included the otherwise indistinguishable contributions from soil and dust.

Rabinowitz [8] used stable lead isotope ratios to evaluate the environments of 3 lead-poisoned Boston children in the early 1980's. Lead isotopes and concentrations were studied in blood and fecal samples, and in air, soil, dust, and paint. He concludes that "In each case, the isotopic composition (IC) of the child's blood lead was identical with the IC of lead paint taken from the child's residence at a site accessible to the child. Fecal lead samples were also identical to that particular paint. Soil lead IC did not always match the IC of local paints. ... Dust in homes that never had lead paint contained lead that resembled lead in urban soils. Dust lead IC did not necessarily have the same IC as current [1981] automobile lead emissions, but appeared to reflect the long-term accumulation of several sources of urban lead fallout. ... These findings may not be directly representative of the sources of lead among children with lower but still excessive blood lead levels (i.e., in the range of 15-30 μg/dl), The lead in the dust in their homes appears to be coming from a large reservoir in the urban soil, which has accumulated over many decades of using lead additives in gasoline ..."

PATHWAYS OF LEAD EXPOSURE

A number of authors have developed statistical models for total lead exposure, including important behavioral variables such as the relative frequency of child mouthing behavior or eating non-food items. These were discussed in detail in EPA's 1986 document on Air Quality Criteria for Lead [9]. Lead paint was banned for use in housing by the 1971 Lead-Based Paint Poisoning Prevention Act. For most children in the U.S., residual lead in LBP and subsequent contamination of soil and household dust are the most important remaining sources of environmental lead exposure [10]. Intact LBP surfaces will produce fine particles by "chalking", which is intentional. Deteriorating LBP surfaces can contribute much larger quantities of particles to soil and household dust while flaking, peeling, or chipping.

While LBP remains a significant potential source of lead exposure if left in place, its improper removal may be at least as hazardous. A number of studies [4,11] have shown that when LBP is removed by older "conventional" methods such as scraping, sanding, or burning, there is a large transient increase in the surface loading of leaded dusts on nearby floors and windows. The large increase in dust lead loading produces substantial increases in blood lead levels in adults, children, and household pets who live in the dwelling unit, and cases of symptomatic lead toxicity are not uncommon. For this reason, most epidemiologic studies of residential lead exposure now ask whether there has been any recent removal of paint or refinishing of furniture on the premises.

The conceptual pathways for childhood lead exposure from LBP are thus well defined. When epidemiologic data are available for measuring exposure (lead in LBP, soil, dust, air, and recent LBP removal), external burden (lead on hands), internal burden (lead in blood), and mitigating behavioral variables (mouthing behavior) or surrogate demographic variables for behavior (age, socio-economic status, household income), multivariate statistical methods may be used to estimate the importance of each of the pathways from LBP to blood lead.

STATISTICAL MODELS FOR CHILD BLOOD LEAD

Many statistical models have been proposed for relating blood lead concentrations (denoted PbBlood) to environmental lead levels (denoted PbPaint, PbSoil, PbDust, PbAir etc.). The environmental lead levels may be either concentrations or surface loadings, depending on available data, and studies are in progress to determine which of the field measurements are the most predictive of blood lead levels. A review of statistical modelling approaches in EPA's Air Quality Criteria for Lead [9] suggested that the following issues must be addressed in any modeling exercise:

(1) lead exposures from different media are additive, and total lead exposure and lead intake should be expressed as the sum of contributions from each medium;
(2) blood lead levels at low to moderate levels of exposure (total lead intake) are approximately linear functions of intake from each medium, but the coefficients reflect differences in the quantity of medium ingested, the availability of lead in ingestion media (soil, dust, paint, food) for dissolution in the stomach, and the absorption or uptake of lead at the entry portal (gut or lung);
(3) at higher levels of lead intake, blood lead levels show less than linear increases with increasing exposure, reflecting biokinetic factors such as limited binding of lead to red blood cells and lower absorption of lead from the gut;
(4) in most epidemiologic studies, the distribution of both environmental lead levels and blood lead levels is highly skewed and may be approximated by a log-normal distribution.

These issues suggested that a consistent method for estimating the contribution of different media would use the following equation for relating blood lead to environmental lead:

$$\text{PbBlood} = B_0 + B_1\,\text{ExteriorPbPaint} + B_2\,\text{Interior PbPaint} + B_3\,\text{PbSoil} + B_4\,\text{PbDust} + \text{etc.} \qquad \text{(Equation 1)}$$

The actual estimation of parameters would be carried out using a logarithmic transformation of both sides of Equation 1:

$$\ln(\text{PbBlood}) = \ln(B_0 + B_1\,\text{ExteriorPbPaint} + B_2\,\text{Interior PbPaint} + B_3\,\text{PbSoil} + B_4\,\text{PbDust} + \text{etc.}) + \text{residual} \qquad \text{(Equation 2)}$$

The use of Equation 2 in estimation implies that the estimand is the geometric mean blood lead level. Note that Equation 2 is intrinsically nonlinear in its parameters B_0, B_1 etc., even though it expresses a linear relationship between biological and physical variables, and so must be fitted using nonlinear regression techniques. Examination of regression residuals shows that Equation 2 both normalizes the residuals and stabilizes their variances, which are desirable statistical properties.

STATISTICAL ANALYSIS OF MULTI-MEDIA PATHWAY MODELS

Unfortunately, Equations 1 or 2 do not allow estimation of the effects of different primary sources such as LBP on blood lead, since they combine both the direct effects of LBP ingestion with the indirect effects of ingesting soil and dust contaminated by LBP. These may be expressed by additional equations for PbDust and PbSoil, similar to Equation 2:

$$\ln(\text{PbDust}) = \ln(D_0 + D_1\,\text{PbSoil} + D_2\,\text{Interior PbPaint} + \text{etc.}) \qquad \text{(Equation 3)}$$

$$\ln(\text{PbSoil}) = \ln(S_0 + S_1\,\text{Exterior PbPaint} + \text{etc.}) \qquad \text{(Equation 4)}$$

Thus the total effect of exterior LBP is not simply B_1, but is:

$$\text{Total Exterior LBP effect} = B_1 + B_3\,S_1 + B_4\,D_1\,S_1 \qquad \text{(Equation 5)}$$

Equations 2, 3 and 4 should not be fitted individually, since the output of Equation 4 is defined as an input variable for Equations 2 and 3, etc. The statistical problems inherent in fitting coupled systems of

equations was first used in studies on environmental lead by R. Bornschein, S. Clark, K. Dietrich, P. Succop and their co-investigators at the University of Cincinnati [12-15]. We have used somewhat different specifications of the SEM than these investigators, in order to account for the biological and physical processes discussed previously in deriving Equations 1 and 2. Equation 1 and related linear models were fitted without transformation using the asymptotically distribution-free AGLS procedure in the EQS program [16]. Equations 2, 3, 4 and related log-transformed linear models were fitted using the program PROC MODEL in the SAS/ETS statistical system [17]. Other analyses of lead data are described by Menton et al. [18].

We will discuss three examples of structural equation modelling (SEM). The first example is an analysis of the data collected in 1989 by the University of Cincinnati investigators around the lead-contaminated community of Midvale, Utah. The second example looks at a cross-sectional study carried out in the active lead smelter community of East Helena, MT in 1983. The third example looks at the study in Butte. MT, carried out by the University of Cincinnati investigators.

DATA FROM THREE CROSS-SECTIONAL STUDIES

These data were provided to us by the Principal Investigator, Dr. Robert Bornschein. Midvale is on the outskirts of Salt Lake City. Metal processing operations have occurred at Midvale for over a century, producing large tailings piles at the edge of the town. Family recruitment, environmental sampling, blood lead and behavioral data collection, and preliminary statistical analyses are reported in [15]. Additional analyses were carried out using methods described above [19,20]. The available data set includes information for 166 children in 128 families.

The other data sets contain similar variables, but are not quite identical. The study at East Helena, Montana, in 1983 was carried out by EPA, CDC, state and local health agencies. The data set used here was that available for the 1989 Office of Air Quality Planning and Standards (OAQPS) staff paper on lead exposure analysis [21,22], supplemented by some additional child and family socio-demographic data. There were some problems in dust measurement from vacuum cleaner samples, with many missing values. These were imputed for this analysis from air and soil lead. The information on lead-based paint was quantitatively uncertain and was replaced by a dummy variable that indicated either presence or absence of lead-based paint anywhere in the house. There was no variable that was directly comparable to the socio-economic status variable (SES) used in the Midvale and Butte studies, so a dummy variable to indicate family income > $15,000 was used. Also, air lead was an important contributor to lead in blood and in soil and dust media, since there was and is an active primary lead smelter at the south edge of the community. These differences illustrate some of the problems in the naive comparison of lead regression coefficients across different studies.

The 1990 Butte study was also carried out by the Univ. of Cincinnati investigators [23] and is in general very similar to the Midvale study, with similar variables. Butte, MT, has been the site of lead mining and smelting activities for over a century. Although these activities are currently inactive, mine waste piles dominate some parts of the community. These analyses are for children whose residences were inside the area closest to the largest mine waste piles. The data set included XRF measurements for both exterior and interior paint.

RESULTS

The results for three very similar analyses at different sites are shown in Table 1, and graphically in Figures 1, 2, and 3. It is seen that there are statistically significant pathways from paint to soil lead to blood lead at all three sites, but that other factors differ from one place to another. The total effect on blood lead of lead in soil or dust for a typical child is the sum of the coefficients of the coefficient for that medium and the coefficient for the product of concentration and mouthing or ingestion of non-food objects. Thus, from Table 1, the blood lead equation for East Helena has terms

1.30* dust lead + 0.73 * dust lead * (Mouthing Score) =2.03 * dust lead * { 0.64 + 0.36 * (Mouthing Score)}.

TABLE 1. REGRESSION COEFFICIENTS FOR BLOOD LEAD, DUST LEAD AND SOIL LEAD EQUATIONS IN STRUCTURAL EQUATION MODELS FOR THREE WESTERN COMMUNITIES. ASYMPTOTIC STANDARD ERRORS IN PARENTHESES.

EQUATION:	BLOOD(μg/dL)			DUST (1000 μg/g)			SOIL (1000 μg/g)		
STUDY:	EAST HELENA	MIDVALE	BUTTE	EAST HELENA	MIDVALE	BUTTE	EAST HELENA	MIDVALE	BUTTE
DUST LEAD (1000 μg/g)	1.30*** (0.41)	0^{con}	0^{con}						
SOIL LEAD (1000 μg/g)	0^{con}	0.44 (0.77)	0^{con}	0.815*** (0.117)	0.546*** (0.084)	0.380*** (0.055)			
AIR LEAD (μg/m^3)	0.76** (0.33)	NA	NA	0.361*** (0.040)	NA	NA	0.35*** (0.04)	NA	NA
LEAD PAINT ON HOUSE (0 or 1)				0.116$^+$ (0.081)	NA	NA	0.23* (0.11)	NA	NA
XRFI (mg/cm^2)				NA	0.002 (0.008)	0.012 (0.034)			
XRFX (mg/cm^2)							NA	0.27* (0.13)	0.070* (0.04)
PAINT REMOVAL (0 or 1)	NA	0.67$^+$ (0.49)	NA	NA	-0.016 (0.038)	NA	NA	-0.192*** (0.052)	NA
DUST LEAD * MOUTHING NON-FOOD (1000 μg/g)	0.73* (0.40)	0^{con}	0^{con}						
SOIL LEAD * MOUTHING NON-FOOD (1000 ppm)	0^{con}	2.12** (0.90)	0.65** (0.24)						
MOUTHING NON-FOOD (Median=1)	0^{con}	0.00 ((0.06)	0.03 (0.32)						
SES (0 TO 1)	NA	-8.9*** (1.7)	-3.1*** (1.26)	NA	-0.257* (0.127)	0.047 (0.210)	NA	-0.750* (0.304)	0.12 (0.21)
HIGH INCOME (0 or 1)	-1.00* (0.60)	NA	NA	-0.069$^+$ (0.048)	NA	NA	-0.11 (0.052)	NA	NA
POST WW2 (0 or 1)							NA	-0.12 (0.20)	NA
HOUSE AGE (100 YEARS)							NA	NA	0.47 (0.46)

Code for statistical significance for one-tailed tests: + = 0.05<P<0.10; *=0.01<P<0.05; **= 0.001<P<0.01; ***= P<0.001; con= Estimate constrained to be non-negative; NA= Not Available; Blank cells indicate no direct pathway (see Figures 1-3).

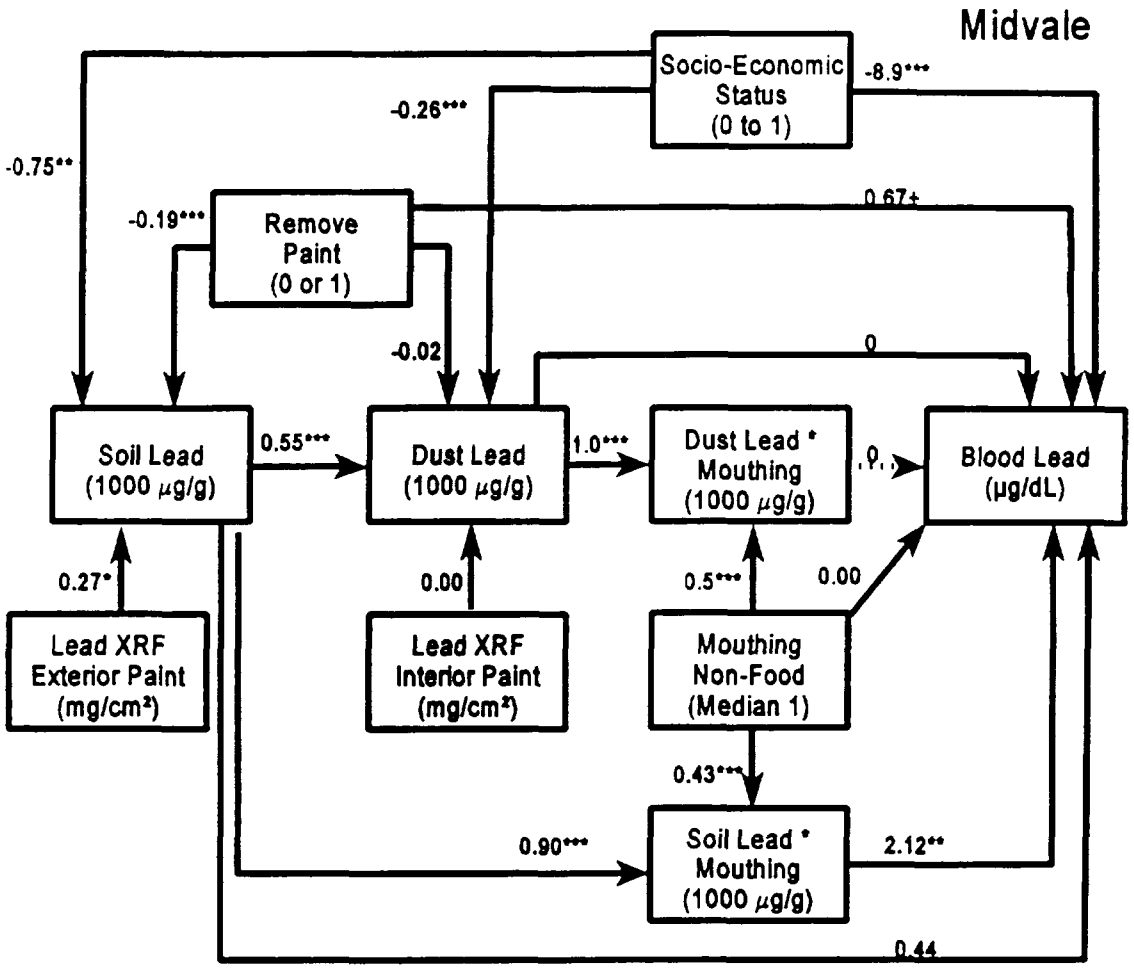

Figure 1. Environmental lead pathways for 1989 Midvale Study. Code for statistical significance: + = 0.05<P<0.10; * = 0.01<P<0.05; ** = 0.001<P<0.01; *** = P<0.001.

Since the typical child has standardized mouthing score = 1, the total effect is 2.03 ug/dl blood lead per 1000 ppm lead in dust. The coefficients for soil lead are 0 when constrained to non-negative values. Thus, for East Helena, air lead and dust lead are significant direct pathways.

The indirect pathways for the East Helena study are important. There is a strong statistically significant dependence of dust lead on both air lead and soil lead. There is a marginally significant relationship of lead-based paint on household dust lead (one-tailed $P < 0.10$). However, the relationship of soil to lead-based paint is statistically significant, about 230 ppm additional lead in soil for houses with lead paint above 1 mg/cm^2. This is then readily transferred from soil to house dust to the child. It is interesting that an independent effect of lead-based paint on blood lead can be detected even when there a great deal of lead exposure from airborne sources.

The Midvale and Butte community studies did not have high air lead concentrations. There are some differences in the relative importance of environmental pathways among the three communities. In the Midvale

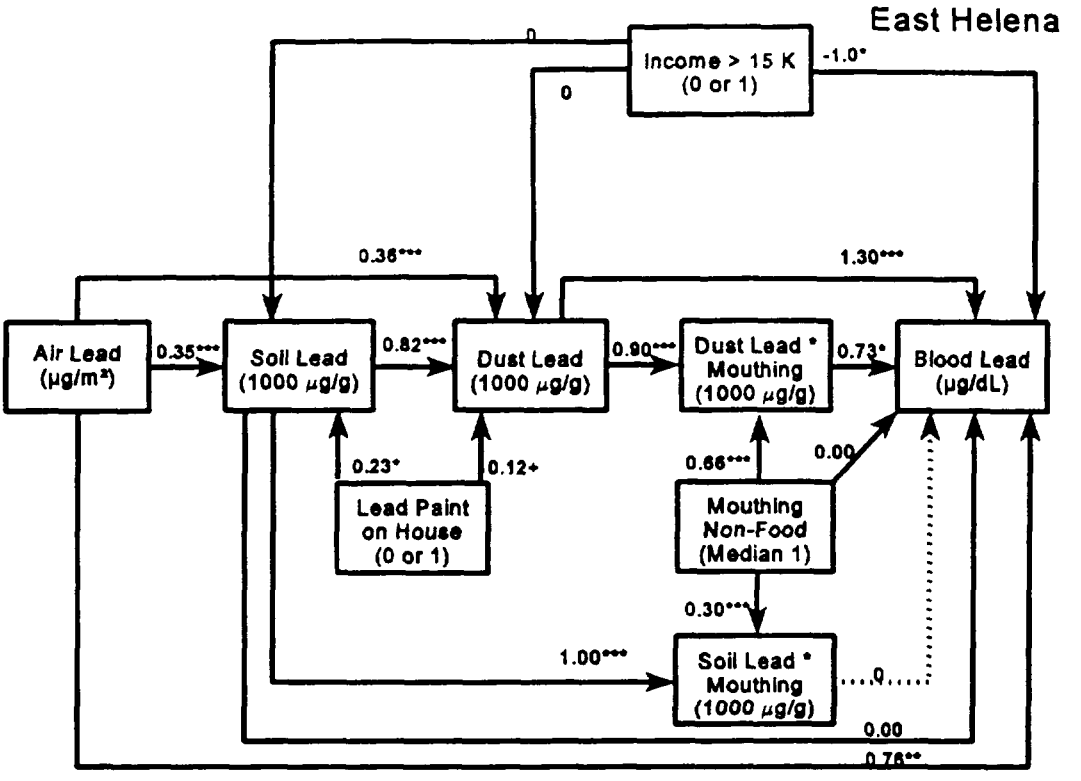

Figure 2. Environmental lead pathways for 1983 East Helena Study. Code for statistical significance: + = 0.05<P<0.10; * = 0.01<P<0.05; ** = 0.001<P<0.01; *** = P<0.001.

study, the direct soil lead pathway is not statistically significant, but the product of soil lead and standardized frequency of mouthing of non-food objects is highly significant. The combined soil lead effect is given by:

0.44 * soil lead + 2.12 * soil lead * (Mouthing Score) = 2.56 * soil lead * { 0.16 + 0.84 * (Mouthing Score)}.

Interior lead-based paint is not significantly correlated with household dust in this study, nor is household dust correlated with blood lead. Perhaps the dust effect is subsumed by the direct and indirect soil lead effect. Recent removal of lead-based paint is marginally associated with a 0.67 μg/dL increase in blood lead. However, there is a very strong statistical relationship between soil lead and exterior lead-based paint (270 μg/g per mg/cm^2) and between soil lead and recent paint removal (190 μg/g less in soil near house with recent paint removal). The effects of socio-demographic factors such as SES and house age may also account for the disappearance of an apparent dust lead effect, which dust lead is confounded with SES and house age.

The results of the Butte study are also shown in Table 1. In the Butte study, the direct soil lead pathway is also not statistically significant, but the product of soil lead and standardized occurrence of mouthing of non-food objects is highly significant. The combined soil lead effect is given by:

0.00 * soil lead + 0.65 * soil lead * (Mouthing Score) =0.65 * soil lead * { 0.00 + 1.00 * (Mouthing Score)}.

Interior lead-based paint is not significantly correlated with household dust in this study, nor is household dust correlated with blood lead. Perhaps the dust effect is subsumed by the direct and indirect soil lead effect. There is a statistical relationship between soil lead and exterior lead-based paint (70 μg/g per mg/cm^2) and between soil lead and dust lead. The effects of socio-demographic factors such as SES and house age may also account for the disappearance of an apparent dust lead effect.

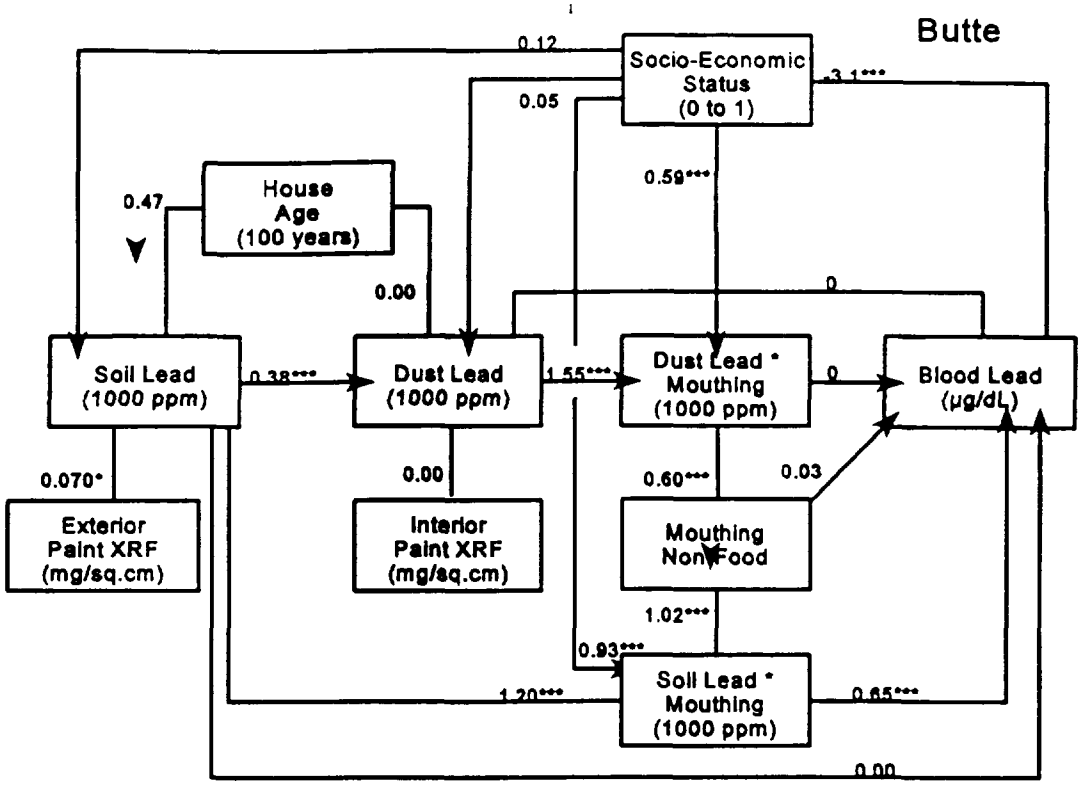

Figure 3. Environmental lead pathways for 1990 Butte Study. Code for statistical significance: + = 0.05<P<0.10; * = 0.01<P<0.05; ** = 0.001<P<0.01; *** = P<0.001.

The relationship of soil lead concentration to dust lead concentration is strong and highly significant in all three studies, largest at an active lead smelter site (0.82 at East Helena), smaller at an inactive lead smelter site (0.55 at Midvale), and smallest -- but not small -- at an inactive lead mining and smelting site (0.38 at Butte). The relationship of blood lead to the most predictive medium is about the same at East Helena and Midvale, 2.03 and 2.56 μg/dL per 1000 μg/g respectively.

DISCUSSION

Lead-based paint is a source of lead in soil in residential yards near houses, whether or not air lead point sources are present. Lead from exterior lead-based paint may thus enter the house as the soil contribution to household dust. Recent studies in other small communities and in urban areas have found quantitatively similar relationships.

The relationship of interior lead-based paint to household dust is harder to detect. The large quantity of paint on the exterior surfaces of an older house with lead-based paint (ca. 8 kg) weathers fairly rapidly and is deposited on the soil near the house. Interior paint deteriorates more slowly and may not be a quantitatively significant component of interior dust, except perhaps in wet rooms such as the kitchen or bathroom. However, detailed studies of individual lead-poisoned children have often found deteriorating lead-painted surfaces nearby, so one must assume that interior lead-based paint can also be a significant source of childhood lead exposure in some cases, even if it is not the most significant source of lead in household dust.

The relationship between blood lead and dust lead or soil lead concentration has two components. The first is the blood lead attributable to dust lead or soil lead without adjustment for mouthing behavior, and the second is the blood lead attributable to the product of dust lead or soil lead and mouthing behavior. The

mouthing behavior variables are different among the studies, and have been normalized to mean value = 1 for comparison. These two components vary considerably in relative importance, with substantial differences between communities. In the East Helena study, the larger coefficient for dust lead was connected to the dust lead main effect (1.30 μg/dL, not to the dust lead * mouthing score interaction term (0.73 μg/dL). In the Midvale study, the estimated regression coefficient on soil lead being 0.44 µg/dl per 1000 ppm, but the regression coefficient on soil lead times mouthing frequency being 2.12 µg/dl per 1000 µg/g in Midvale. Children with frequent ingestion or mouthing of non-food items would show an even larger response.

The income level variable is much less predictive in the 1983 studies than was the SES variable used in the 1989-1990 studies. We suspect that parental education and household hygiene practices that affect infant and toddler lead exposure and nutrition are described better by SES than by income. There is an increase in blood lead after age 6 to 12 months.

These studies also confirmed that even in communities where lead deposition from historical mining and smelter activities is a very significant source of lead contamination of soil, there is also a detectable contribution to soil lead from exterior lead-based paint. Direct evidence of the importance of the exterior lead paint to soil to blood pathway was provided by stable lead isotope studies [7,8]. The apparent lack of a detectable pathway from interior lead-based paint to household dust requires further study.

It is obvious that some of the most highly elevated child blood leads in these communities are associated with the ingestion of deteriorating lead paint, but some of the other elevated blood leads are associated with elevated dust lead concentrations when XRF levels are low. Thus, ingestion of non-LBP components of soil and dust must also be substantial.

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